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5	0	((hepatic adj cell)near3 (transformed or transfected or transform or transfect or transforming or transfecting or transformation or transfection)) near5 p450	USPAT; US-PGPUB; EPO; JPO; DERWENT	2004/01/20 18:28
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AN   1998:494568  BIOSIS
DN   PREV199800494568
TI   Regulation of hepatic cytochrome P450 2C11 by
     transforming growth factor-beta, hepatocyte growth
     factor, and interleukin-11.
AU   Iber, Heinrich; Morgan, Edward T. [Reprint author]
CS   Dep. Pharmacol., Emory Univ., Atlanta, GA 30322, USA
SO   Drug Metabolism and Disposition, (Oct., 1998) Vol. 26, No. 10, pp.
     1042-1044. print.
     CODEN: DMDSAI. ISSN: 0090-9556.
DT   Article
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LA English
ED Entered STN: 18 Nov 1998
Last Updated on STN: 18 Nov 1998
AB Injection of rats with bacterial lipopolysaccharide down-regulates P450 (P450) 2C11 (2C11) mRNA to about 20% of its control levels after only 6 hr, and this level is maintained for at least 48 hr. Although we and others have demonstrated that this effect may be at least partially mediated by the cytokines interleukin-1, interleukin-6, and tumor necrosis factor-alpha, as well as by glucocorticoids, the time courses and potencies of 2C11 repression by each single mediator suggested that no cytokine alone is responsible for the entire time course of 2C11 suppression during inflammation. Here, we show that transforming growth factor-beta, hepatocyte growth factor, and interleukin-11 are potent inhibitors of 2C11 expression. In all three cases, 0.1 ng/ml was enough to down-regulate 2C11 mRNA levels to 50% of control. Interleukin-8, a cytokine that is secreted during the acute phase response but does not influence the liver acute phase response, did not affect 2C11 expression. The various mediators have different time courses of 2C11 down-regulation, indicating that the roles of each may be different at different phases of the response.

L4 ANSWER 2 OF 2 CANCERLIT on STN DUPLICATE 2
AN 1999382921 CANCERLIT
DN 99382921 PubMed ID: 10453545
TI Transformation of rat hepatocytes in an in vitro primary culture by aflatoxin B1.
AU Yuan B; Sun Z
CS Cancer Institute, CAMS and PUMC, Beijing.
SO CHUNG-KUO I HSUEH KO HSUEH YUAN HSUEH PAO ACTA ACADEMIAE MEDICINAE SINICAE, (1997 Feb) 19 (1) 6-10.
Journal code: 8006230. ISSN: 1000-503X.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA Chinese
FS MEDLINE; Priority Journals
OS MEDLINE 1999382921
EM 199910
ED Entered STN: 19991112
Last Updated on STN: 19991112
AB Aflatoxin B1 (AFB1) is one of the major causative factors of hepatocellular carcinoma. In this study, the combined effects of AFB1 activated by human cytochrome **p450** IA2 and c-myc in **transformation** of rat **hepatocytes** were investigated in an in vitro primary culture system. The expression vectors, Xm-6/c-myc was first constructed and their expression possibilities were examined in Alexander cells by immunocytochemistry. Then both c-myc and human cytochrome p450 IA2 expression vectors were sequentially transfected into newborn rat liver cells in serum-free primary culture. Results showed that p450 IA2 could activate AFB1 at concentrations as low as 5 ng/ml, and the activated AFB1 coupled with exogenous c-myc could induce rat hepatocytes to survive and grow beyond two-month limit in primary culture. During long-term in vitro culturing including four-month in crisis, one of the randomly selected transformed hepatocytes with the growth advantage became immortalized. Immunocytochemical assays for CK-18 and rat albumin plus observed electron microscopic features clearly confirmed these cells derived from epithelial hepatocytes. Further characterization showed that the process of immortalization was associated with chromosomal abnormalities and elevated expression of TGF alpha.

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